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Epidemiologic and clinical aspects of feline immunodeficiency virus infection in cats from the continental United States and Canada and possible mode of transmission

Janet K. Yamamoto, PhD; Herman Hansen, DVM; Esther W. Ho, BS; Teresa Y. Morishita, MS; Takiko Okuda, MS, DVM; Thomas R. Sawa, DVM, PhD; Robert M. Nakamura, DVM, PhD; Niels C. Pedersen, DVM, PhD

Summary: *The epidemiologic features of feline immunodeficiency virus (FIV) infection were evaluated in 2,765 cats from the United States and Canada. Of these cats, 2,254 were considered by veterinarians to be at high risk for the infection, and 511 were healthy cats considered to be at low or unknown risk. Of the cats in the high-risk group, 318 (14%) were found to be infected with FIV. The infection rate among low- or unknown-risk cats was 6 of 511 (1.2%). Male cats in the high-risk group were 3 times more likely to be infected than were females, similarly as were cats >6 years old, compared with younger cats; domestic cats, compared with purebred cats; and free-roaming cats, compared with confined cats. Feline immunodeficiency virus and FeLV infections did not appear to be linked with each other; 16% of FeLV-infected cats in the high- and low-risk groups were coinfected with FIV. In contrast, there was a pronounced linkage between FIV and feline syncytium-forming virus (FeSFV) infections. Seventy-four percent of FeSFV-infected cats in the high-risk study group were coinfected with FIV, compared with a 38% FIV infection rate among cats that were not infected with FeSFV.*

The major clinical manifestations associated with FIV infection in cats that were surveyed included chronic oral cavity infections (56%), chronic upper respiratory tract disease (34%), chronic enteritis (19%), and chronic conjunctivitis (11%). Bacterial infections of the urinary tract (cystitis), skin, and ears were seen in a small proportion of cats. Fever of unspecified origin, anorexia, weight loss, lethargy, vomiting, abnormal behavioral problems, lymphosarcoma, and myeloproliferative disorders were seen as accompanying or primary features of the disease in a proportion of affected cats. Anemia and leukopenia were observed in about a third of the cats that were clinically ill at the time of testing.

Experimental studies indicated that biting was a highly efficient mode of transmission. Cats with clinical signs of illness tended to shed more virus in their saliva than did healthy carrier cats. Seroepidemiologic studies also supported the notion that territorial aggression and biting were important natural modes of FIV transmission.

A group of 18 human beings who had intimate contact with FIV-infected cats were found to be seronegative for the virus. There was no positive evidence that FIV was of public health importance.

In 1986, a T-lymphotropic lentivirus initially was isolated from a group of cats with an acquired immunodeficiency syndrome-like disease.[1] An identical virus was detected in cats from Japan[2] and the United Kingdom[3] and was described in 1988. In the years since the discovery of the virus, diagnostic tests have been developed to detect the infection, and veterinarians throughout the world have begun to test an increasing number of cats.

The causative agent of this newly recognized disease syndrome is a lentivirus that is morphologically and biochemically similar to human immunodeficiency virus, but is antigenically distinct.[1] The virus originally was named feline T-lymphotropic lentivirus, which was descriptive of a major feature of the virus, ie, growth in T lymphocytes, and alluded to the taxonomy of the agent. Now that it is clearly established that the virus is a major cause of a chronic acquired immunodeficiency-like syndrome in cats, the virus has been renamed feline immunodeficiency virus (FIV).[4] This name reflects its taxonomic and pathogenic relationship to human and simian immunodeficiency viruses (HIV, SIV). The names HIV, SIV, and FIV are in accordance with the nomenclature selected for this type of virus by an international committee.[5]

Animal inoculation studies have identified the primary stage of infection.[1,6] Within 4 to 6 weeks of inoculation, infected cats developed transient fever, leukopenia, and neutropenia lasting several weeks.

From the Department of Medicine, School of Veterinary Medicine, University of California, Davis, CA 95616 (Yamamoto, Ho, Morishita, Okuda, Pedersen), Veterinary Laboratory Branch, State of Hawaii, Department of Agriculture, Division of Animal Industry, 99-762 Moanalua Rd, Area, HI 96701 (Sawa), Department of Animal Science, University of Hawaii-Manoa, Honolulu, HI, 96822 (Nakamura), and Hansen's Veterinary Immunology, 450 Porter, Ste C, Dixon, CA 95620 (Hansen).

This study was supported in part by NIAID-1-RO1-AI25802-01, Public Health Services grants CA-39016, Save Our Cats and Kittens (SOCK) Inc, the Winn Foundation, Cat Fanciers Association, and the Ralston-Purina Corporation.

This phase of the infection was associated with generalized lymphadenopathy that lasted many months before subsiding. At the present time, however, none of the specific pathogen-free (SPF) cats that were infected with the virus have entered the terminal phase of illness.[6] The virus appears to be transmitted with great difficulty by intimate contact alone.[6] In utero and neonatal (lactogenic) infections also have not been observed under experimental conditions.[6] The virus is transmitted readily by parenteral inoculation of blood and plasma, and is detected in high concentration in saliva.[6] Bites were postulated as one possible mode of transmission.[6]

Other than initial descriptions of the naturally acquired disease,[1-3] there is little information concerning the spectrum of illnesses that can be associated with FIV infection. The predominant disease syndrome consists of chronic infections of the mouth (periodontitis, stomatitis), nasal passages, conjunctiva, ears, skin, and intestinal tract. This immunodeficiency-like syndrome progressively worsens over months or years. Behavioral abnormalities, apparently associated with CNS involvement, have been observed in some infected cats.[1,3] Hematologic abnormalities, usually leukopenia and anemia, have been seen in a proportion of infected cats.[1,3,6] A myeloproliferative disorder developed in one cat with experimentally induced FIV infection.[1,6]

The epidemiologic features of FIV infection in cats have not been well studied. The virus is virtually worldwide in distribution,[1-3] but risk groups have not been identified. There is a suggestion, however, that free-roaming (indoor/outdoor, stray, feral) cats are at higher risk, and that bringing cats together from such environments may lead to localized epizootics.[1]

The public health hazards of FIV have been minimized on the basis of preliminary reports.[1] The virus is antigenically distinct from HIV, and several human beings in prolonged and intimate contact with infected cats did not have serologic evidence of FIV infection.[1] The high species-specificity of various lentivirus isolates also has tended to assure the public that cats are not a source of human disease. Nevertheless, more serologic studies in this area remain to be done.

The study reported here was derived from a large seroepidemiologic survey of 2,765 cats in North America. Of these cats, 2,254 had clinical signs of disease or exposure history suspicious enough to lead veterinarians to request testing, and 511 were healthy-appearing cats considered to be at low risk. In all, 324 of 2,765 cats were found to be infected.

This study is concerned mainly with data collected from these cats. The information included age, gender, major clinical signs of disease, hematologic abnormalities, and incidence of exposure to 2 other retrovirus infections of cats (feline syncytium-forming virus [FeSFV] and FeLV). Experimental studies pertaining to the likely mode of transmission of the virus in nature, risk groups, and lack of cat-to-human transmission also were evaluated.

Materials and methods

Serologic survey—A serologic survey of 2,254 domestic cats from a high-risk group of cats in the continental United States and Canada was performed. The cats of this group had clinical signs of disease that were typical of those described for FIV-infected cats in a diseased cattery[1] or that were in contact with such infected cats. An additional group of sera was obtained from 511 cats considered to be at low or unknown risk for FIV infection. Cats of the low- or unknown-risk group were pets and were from the continental United States and Canada (n = 352), were feral from the campus of the University of California at Davis (n = 77),[a] or were from the Yolo County pound or San Francisco Society for the Prevention of Cruelty to Animals shelter (n = 82).[a] Veterinarians submitted serum samples (1 to 2 ml) along with negligible to detailed clinical and hematologic histories. The collected sera were tested for FIV antibodies by indirect fluorescent antibody (IFA) assay or by immunoblot analysis. A portion of the collected sera also was tested at random for FeLV by ELISA for FeLV-p27 antigen and for FeSFV antibodies[7] by IFA assay on FeSFV-infected *Felis catus* whole fetus-4 cells (Fcwf-4).[8]

Indirect fluorescent antibody assay for FIV antibodies—Sera were tested for FIV antibodies by IFA assay, using FIV-infected blood lymphocytes or infected Crandell feline kidney cells as substrates.[1,6] The sera were tested at a dilution of 1:10 in phosphate-buffered saline solution (PBSS). Fifteen microliters of diluted sera was placed on individual wells of acetone-fixed IFA slides of FIV-infected cells, which were incubated for 30 minutes at 37 C in a moist chamber. The slides were washed 3 times in PBSS and then were incubated for an additional 30 minutes at 37 C with fluorescein-conjugated rabbit anti-cat IgG.[b] After washing, the slides were counterstained with 0.01% Evans blue, mounted in 50% glycerol in PBSS, and read by use of a fluorescent microscope. The serum samples were considered positive for FIV antibodies when giant cells and infected cells had typical membrane and cytoplasmic fluorescence. Positive and negative control sera were included in every assay. For further confirmation of test sensitivity, samples of IFA-positive and IFA-negative control sera also were tested at random by immunoblot analysis. Any serum that was inconclusively positive by IFA was automatically retested by immunoblot analysis.

[a]Serum samples were obtained from these cats as part of a feral cat neutering and disease control study conducted on the campus of the University of California at Davis. The project leaders were Drs. Julie Levy and Kathy James.

[b]Antibodies Inc, Davis, Calif.

Immunoblot analysis—The virus from FIV-infected cell culture fluid was clarified by low-speed centrifugation, concentrated by ultracentrifugation, and purified by passage over a 10%/50% (w/v) continuous sucrose gradient.[1] Virus purified by this procedure was electrophoretically transblotted onto nitrocellulose paper as the substrate for the immunoblots.[1,6] Serum samples were diluted 1:50 in buffer 3 (0.15*M* sodium chloride, 0.001*M* edetic acid, 0.05*M* Tris base, 0.05% Tween 20, and 0.1% bovine serum albumin) and were incubated with virus blot strips in separate glass tubes for 12 hours at 37 C. The blot strips were washed individually and incubated for 1 hour at 37 C with peroxidase-conjugated rabbit anti-cat IgG[c] for cat serum and peroxidase-conjugated rabbit anti-human IgG[c] for human serum. After extensive washing, each strip was incubated with a fresh substrate solution (0.05% diaminobenzidine and 0.01% H_2O_2 in 0.1*M* Tris, pH 7.4) for 4 to 10 minutes at 20 C. The reaction was stopped by addition of excess distilled H_2O when visible bands had been established, and the strips were blot-dried. The molecular weight of each band on the immunoblots then was determined by comparing the migration distance of the molecular standards on the strip previously stained with amido black. Positive and negative control sera were included in each immunoblot analysis as internal controls for diagnostic evaluation.

Isolation of FIV from infected lymphocytes and saliva—Blood mononuclear cells (BMC) obtained from SPF cats were purified by ficoll-diatrizoate sodium gradient centrifugation,[9] and were stimulated with 5 μg of concanavalin A/ml for 3 days in a 5% CO_2 incubator at 37 C. The cells were washed with Hanks balanced salt solution, resuspended in RPMI 1640 culture medium at a final concentration of 0.5 to 1×10^6 cells/ml, and placed in 25-cm^2 culture flasks. These uninfected, stimulated BMC were cocultivated at 1:1 with infected BMC prepared by the same method, or cultured with 0.2 to 0.5 ml of saliva. Culture media consisted of RPMI 1640, 10% heat-inactivated fetal bovine serum, 10 mM HEPES, penicillin (100 U/ml), streptomycin (10 μg/ml), gentamicin (50 μg/ml), 2 mM L-glutamine, 5×10^{-5}*M* 2-mercaptoethanol, polybrene (2 μg/ml), and recombinant human interleukin-2[d] (2,100 U/ml). Cultures were incubated in 5% CO_2 at 37 C and monitored every other day for giant cell formation and cell death. Culture supernatants were harvested for assay for reverse transcriptase (RT) activity, and the cells were resuspended in fresh culture medium on every third day or twice a week. These cultures were further supplemented with stimulated cells from the uninfected BMC stock to maintain a minimal cell concentration of 0.5 to 1.0×10^6 cells/ml. The culture was considered negative for virus infection and was terminated on day 42 of the culture period when the culture indicated lack of RT activity, and cells were IFA-negative when exposed to FIV-positive serum. The RT assay used poly(rA)-oligo(dT_{12-18}) as template primer, 5 μCi[^{3}H]-labeled thymidine triphosphate/sample, and Mg^{2+} as divalent cation for detecting FIV.[1,6,10] The culture fluids also were tested for FeLV-p27 by results of ELISA.[7] The cells were tested for FeSFV infection by IFA; positive control antiserum was produced by inoculating SPF cats with FeSFV[8]

Experimentally induced FIV infection—In earlier studies,[1,6] 8- to 16-week-old SPF kittens had been inoculated IV or intraperitoneally (IP) with 1 to 2 ml of FIV inoculum; kittens 2428 and 2429 had been inoculated IV with infected whole blood or plasma, respectively. For this study, kitten 2841 was inoculated IP with infected cell culture fluid with RT activity of 70,000 counts per minute (cpm)/ml. Kittens J13, J14, and J15 were given multiple inoculations (once a week for 3 weeks) of 1 ml of pelleted virus (FIV isolate from No. 2429), with RT activity of 500,000 cpm/ml. Kitten J13 was inoculated IV, whereas the others were inoculated IP. Kittens were monitored daily for overt clinical signs and weekly by physical examination.[6] Saliva and blood lymphocytes from experimentally infected kittens were tested for FIV isolation. At the time of blood or saliva sample collection, kittens 2428 and 2429 (3 months after infection) had generalized lymphadenopathy, and kittens J13, J14, and J15 (8 months after infection) lacked clinical signs of disease. Blood and saliva samples were obtained from kitten 2841 at the time of death (2 months after infection). This kitten was suffering from pancytopenia and anemia and had a myeloproliferative disorder.[6]

Naturally infected cats—Cats with naturally acquired FIV infection were adults (with clinical signs of disease) that ranged in age from 2 to 6 years. Cats were obtained from areas around Honolulu, Hawaii. Cat CJ1 was a domestic shorthair male with ulcerative plasmacytic stomatitis, gingivitis, periodontitis, and hypersalivation. Cat CJ2 was a domestic longhair female with chronic otitis externa, generalized lymphadenopathy, and no sign of oral disease. Cat CJ3 was a domestic shorthair male with severe gingivitis. Cat CJ4 was a domestic shorthair male with gingivitis, ulcerative plasmacytic stomatitis, periodontal disease, hypersalivation, and chronic ear infections.

Experimental bite study—Two of the cats with naturally acquired FIV infection (No. CJ1 and CJ2) and with clinical signs of the immunodeficiency phase of infection were selected for the study. These cats had high concentration of FIV in their saliva. Four SPF kittens, 16 to 20 weeks old (No. 3326, 3327, 3334, and 3340), were used as bite recipients. Kittens 3326 and 3340 were exposed to cat CJ2, and kittens 3327 and 3334 were exposed to cat

[c]Cappel Laboratories, Cochranville, Pa.
[d]Cetus Corp, Emeryville, Calif.

CJ1. All 6 cats were anesthetized briefly by use of IV administered ketamine and acepromazine. Opposing sites on the medial and lateral aspects of the caudad thigh were shaved, surgically washed, and disinfected with organic iodide. The mouth of the anesthetized donor cat was opened, and the corresponding upper and lower canine teeth were positioned over the surgically prepared area on the bite recipient. The mouth was manually closed, forcing the opposing canine teeth to penetrate the skin and underlying musculature. The bite-recipient kittens then were treated with penicillin for 3 days to prevent development of abscesses at the bite sites.

Statistical methods—The *P* value was derived by testing the difference between 2 rates in a 2-sample *Z* test.[11] The difference in the 2 rates was considered to be significant when the *P* value was <0.05 (Z value >1.96 at $\alpha = 0.025$) at a 95% confidence interval.

Public health studies—Sera from 18 human beings with substantial accidental exposure to FIV or FIV-infected cats were selected for this study. Sera were from practicing veterinarians, cat owners, and professional or technical research staff working with the virus or virus-infected cats in a natural or experimental setting. Sera were tested by immunoblotting for antibodies to gradient-purified FIV as described previously.

Results

Feline immunodeficiency virus serologic survey—The serologic survey of domestic cats in the United States and Canada for FIV antibodies involved 2,254 cats with a medical history of chronic infectious diseases or prolonged and intimate contact with such cats (high-risk group) and 511 healthy-appearing cats with no known exposure to FIV (low-risk group). Approximately 14% (310/2,212) of the high-risk group of cats surveyed in the continental United States and 19% (8/42) of those surveyed in Canada were seropositive for FIV antibodies (Table 1). The 14% (318/2,254) infection rate among high-risk cats from the continental United States and Canada was significantly ($P \leq 0.001$) greater than the 1.2% (6/511) infection rate in cats at low or unknown risk (Table 2).

In the high-risk group, there were 663 females, 855 males, and 736 cats without gender description. Of the 1,518 high-risk, FIV-positive cats with gender description, 51 females and 168 males were seropositive for FIV. Thus, more males than females were infected at a ratio of 3.3:1 (Table 1). Because the male-to-female ratio in the entire high-risk group of cats was 1.3:1 (855:663), the difference was significant ($P \leq 0.001$). The predominance of FIV-infected males in the high-risk group also was significant ($P = 0.024$), compared with the male-to-female ratio of 0.97:1 among cats at low risk.

Cats kept strictly indoors had a significantly ($P \leq 0.0026$) lower incidence of FIV infection than did cats that were kept strictly outdoors (Table 1). This trend also was evident when the incidence of FIV infection in outdoor/indoor and outdoor cats was compared with the incidence of infection in cats that were not allowed outside access, but the difference was not quite significant at the 95% confidence interval. The incidence of FIV infection in the high-risk group of cats was significantly ($P \leq 0.001$) higher in domestic than in purebred

Table 1—Serosurvey of domestic cats from high-risk groups in the United States and Canada

Description	Total surveyed	No. of cats seropositive for FIV (%)
Geographic distribution		
United States	2,212	310 (14%)
Canada	42	8 (19%)
Gender distribution		
F	663	51 (8%)
M	855	168 (20%)
Undescribed gender	736	99 (13%)
Age distribution (yrs)		
<1	198	7 (4%)
1 to 5	678	70 (10%)
6 to 10	390	94 (24%)
11 to 15	205	49 (24%)
>15	19	3 (16%)
Unspecified age	764	95 (12%)
Breed distribution		
Domestic shorthair	449	76 (17%)
Domestic longhair	128	19 (15%)
Siamese	71	4 (6%)
Persian	40	0 (0%)
Himalayan	18	0 (0%)
Burmese	10	0 (0%)
Abyssinian	9	0 (0%)
Main Coon	7	0 (0%)
Others	16	1 (6%)
Unspecified breed	1,506	218 (14%)
Environmental status		
Indoor (owned)	61	5 (8%)
Outdoor (owned)	33	11 (33%)
Indoor/outdoor (owned)	90	18 (20%)
Outdoor (unowned)	28	7 (25%)
Unspecified	2,042	277 (14%)

Table 2—Serosurvey of domestic cats from groups at low risk for feline immunodeficiency virus infection (FIV) in the continental United States and Canada

Vital statistics	Total	FIV infected	FeLV infected	FIV and FeLV infected
Gender distribution				
F	254	0	0	0
M	247	5	5	1
Undescribed gender	10	0	0	0
Age distribution (yr)				
<1	86	0	0	0
1 to 5	157	0	0	0
6 to 10	54	1	0	0
11 to 15	15	1	0	0
>15	2	0	0	0
Adults (age unknown)	197	3	5	1
Breed distribution				
Domestic	473	5	5	1
Purebred	38	0	0	0
Ownership status				
Abandoned, feral, stray	159	3	5	1
Owned	352	2	0	0

Table 3—Coinfection of cats with feline immunodeficiency virus (FIV), FeLV, and feline syncytium-forming virus (FeSFV)

Test description and status	No. of cats tested	No. of cats that were FIV-positive (%)
FeLV-27 antigenemia		
Positive	215	34 (16%)
Negative	1,394	226 (16%)
FeSFV antibody status*		
Positive	116	86 (74%)
Negative	88	33 (38%)

*The presence of antibodies to FeSFV in the serum is correlated completely with persistent infection.[4]

*Table 4—Hematologic abnormalities in feline immunodeficiency virus (FIV)-positive cats**

	Normal ranges	Total surveyed	No. of cats (%) Low	High
PCV (%)	24 to 45	40	12 (30%)	2 (5%)
RBC (10^6 cells/μl)	5 to 10	36	15 (42%)	0
WBC (10^3 cells/μl)	5.5 to 19.5	47	14 (30%)	7 (15%)
Lymphocytes (10^3 cells/μl)	1.5 to 7.0	34	18 (53%)	2 (6%)
Neutrophils (10^3 cells/μl)	2.5 to 12.5	37	8 (22%)	13 (35%)
Platelets (10^3 cells/μl)	300 to 800	25	4 (16%)	1 (4%)

*Only one cat in this survey was seropositive for FeLV.

*Table 5—Clinical signs of disease in feline immunodeficiency virus (FIV)-positive pet cats**

Description	No. of cats (%)
No. of FIV-positive cats with a disease history	193
Oral signs	
Chronic gingivitis	62 (32%)
Chronic periodontal disease	22 (11%)
Chronic stomatitis	15 (8%)
Ulcerations of tonsils and tongue	10 (5%)
Gastrointestinal tract signs	
Chronic diarrhea (enteritis)	34 (18%)
Others (unspecified)	3 (2%)
Respiratory tract diseases	
Chronic rhinitis	45 (23%)
Upper respiratory tract infections (unspecified)	18 (9%)
Others	2 (1%)
Miscellaneous signs	
Weight loss	43 (22%)
Fever	32 (17%)
Anorexia	33 (17%)
Lymphadenopathy	22 (11%)
Chronic conjunctivitis	21 (11%)
Chronic abscess	20 (10%)
Chronic infection	17 (9%)
Dermatitis	15 (8%)
Lethargy	15 (8%)
Vomiting	16 (8%)
Recurrent cystitis	9 (5%)
Neoplasia (lymphosarcoma [n = 3], myeloproliferative diseases [n = 2], mammary tumor [n = 1])	6 (3%)
Otitis	4 (2%)
Abnormal behavior (CNS involvement)	2 (1%)

*Only one cat in this survey was seropositive for FeLV.

cats. Although purebreds made up 22.9% (171/748) of the cats (with breed specified) in the high-risk population, only 3% (5/171) were FIV-infected. Infected cats of the high-risk group were significantly older than cats of the population as a whole; 23.8% (146/614) of cats ≥6 years old were infected with FIV, compared with an 8.8% (77/876) infection rate in cats <6 years old ($P \leq 0.001$).

Among the 215 cats of the high-risk group that were seropositive for FeLV-p27 antigen, 34 (16%) also were infected with FIV (Table 3). Similarly, 16% (226/1,394) of the FeLV-seronegative cats of the same risk group also were infected with FIV. Similar percentages were observed for coinfections (FIV and FeLV) in cats of the low- or unknown-risk group (Table 2). Thus, there appeared to be no significant linkage between FeLV and FIV infections in the 2 population groups that were studied. In contrast, 74% (86/116) of FeSFV-infected cats were coinfected with FIV. This was significantly ($P \leq 0.001$) higher than the 38% (33/88) incidence of FIV infection among FeSFV-negative cats of the same cohort.

Hematologic survey of FIV-positive cats—Participation in the hematologic survey was sparse, and the hematologic status of the cats described in this study was only from the time of initial FIV testing (Table 4). Only one cat (with clinical signs of disease) in this portion of the survey was coinfected with FeLV. Decreased PCV (30%), decreased RBC count (42%), and leukopenia (30%) were frequently observed in FIV-infected cats that had clinical signs of disease. Leukopenia was associated with lymphopenia (53%) and neutropenia (22%). Cats with leukocytosis (15%) usually had neutrophilia (35%). Mild thrombocytopenia was observed in 16% of the FIV-infected cats with clinical signs of disease, but none had thrombocyte count $< 10^5$ platelets/μl.

Clinical manifestations of FIV infection—In the high-risk group, only 193 FIV-seropositive cats had adequate clinical history. Of these, 56% had signs referrable to the oral cavity, 34% to the respiratory tract, and 20% to the gastrointestinal tract (Table 5). Oral disease usually was manifested by variable degrees of gingivitis, periodontitis, and stomatitis. Many of these cats responded initially to supportive treatment, but with time, they became refractory to treatment. The oral disease often progressed to a stage requiring tooth extraction. The cats then became anorectic and often succumbed to a wasting syndrome. Gastrointestinal tract signs included chronic diarrhea, dehydration, and emaciation. A high mortality also was observed in cats with signs of chronic gastrointestinal tract disease. Upper respiratory tract disease consisted of chronic rhinitis with or without conjunctivitis. Nonspecific signs such as fever, anorexia, weight loss, and lethargy were common in cats infected with FIV. Other clinical signs observed in FIV-infected cats were lymphadenopathy, chronic abscesses, and chronic microbial infections of the skin and ears. Anemia often was observed in the terminal stages of the disease.

Feline immunodeficiency virus isolation from blood and saliva—The concentration of virus in BMC and saliva (Table 6) was measured in the 6 experimentally infected kittens (No. 2428, 2429, 2841, J13, J14, J15) and in 4 cats with naturally acquired infection (No. CJ1, CJ2, CJ3, CJ4). Virus concentra-

Table 6—Feline immunodeficiency virus (FIV) isolation from blood lymphocytes (BL) and saliva of FIV-infected cats

Cat No.	Days after infection	Virus source	Disease status	Reverse transcriptase expression in cell cultures infected with BL or saliva (10^3 cpm/ml)* Days after incubation 3	6	9	12	15	18	21	30	45
Experimentally infected												
2428	90	BL;	Healthy	3	3	0.6	0.2	2	0.5	0.5	0.4	8
		saliva		0	0	0.7	0	0.4	0	0	0	0.3
2429	90	BL;	Healthy	0	0	11	56	11	24	13	124	13
		saliva		2	4	0.4	1	0.7	1	0.3	1	32
2841	60	BL;	ill	8	3	3	2	13	68	31	50	ND
		saliva		ND	11	54	35	22	116	70	ND	ND
J13	240	BL;	Healthy	2	1	37	131	181	360	232	ND	ND
		saliva		1	7	83	285	217	235	314	294	300
J14	240	BL;	Healthy	0	2	28	200	178	244	243	ND	ND
		saliva		0	0.9	1	0.4	0.8	3	2	2	0.2
J15	240	BL;	Healthy	0.5	5	98	249	275	302	237	ND	ND
		saliva		0	4	38	211	145	148	189	185	236
Naturally infected												
CJ1	...	BL;	ill	2	1	5	42	127	119	109	94	62
		saliva		2	2	8	8	129	221	267	373	49
CJ2	...	BL;	ill	5	34	85	395	310	468	389	233	76
		saliva		2	3	8	8	108	168	341	430	59
CJ3	...	BL;	ill	0.9	5	83	333	228	185	179	99	123
		saliva		2	3	25	79	407	274	328	387	139
CJ4	...	BL;	ill	3	7	35	182	194	329	209	182	73
		saliva		2	3	3	127	311	328	331	439	284

*The time after incubation when reverse trascriptase appears, and its concentration, are proportional to the amount of virus in the initial inoculum. A large amount of virus in the initial inoculum will lead to a rapid appearance and high reverse transcriptase activity, whereas a small amount of virus will have the opposite effect.

cpm = counts per minute; ND = not determined.

Table 7—Serosurvey of human beings exposed to feline immunodeficiency virus (FIV) or FIV-infected cats

Human being No.	Occupation	Type of exposure	Duration of exposure or time since last exposure	FIV antibody status (by immunoblot analysis)
1	Veterinarian	Owner of FIV-positive cat	2 yr	Negative
2	Veterinarian	Needle stick with infected blood	8 mo later	Negative
3	Veterinarian	Cat bites from FIV-infected cats	1 yr later	Negative
4	Cat owner	Close contact with FIV-positive cat	4 yr	Negative
5	Cat owner	Close contact with FIV-positive cat	4 yr	Negative
6	Cat owner	Close contact with FIV-positive cat	4 yr	Negative
7	Cat owner	Close contact with FIV-positive cat	2 yr	Negative
8	Cat owner	Close contact with FIV-positive cat	1 yr	Negative
9	Cat owner	Close contact with FIV-positive cat	Not stated	Negative
10	Animal handler	Close contact with FIV-positive cat	1 yr	Negative
11	Animal handler	Close contact with FIV-positive cat	7 mo	Negative
12	Animal handler	Close contact with FIV-positive cat	4 mo	Negative
13	Animal handler	Close contact, scratched, bitten	1.5 yr	Negative
14	Researcher	Close contact with infected culture fluids	1 yr	Negative
15	Researcher	Close contact with infected culture fluids	1.5 yr	Negative
16	Researcher	Needle stick with concentrated virus	1.5 yr later	Negative
17	Researcher	Close contact with infected culture fluids	6 mo	Negative
18	Researcher	Contaminated skin wound with infected blood	6 mo later	Negative

tions in the blood and saliva of experimentally infected cats were low during the early stages of infection, but tended to increase after 6 months. Although blood and salivary concentrations of virus tended to be similar, 1 experimentally infected cat (No. CJ14) had low virus concentration in saliva and high virus concentration in blood. There was some correlation between virus concentration in the saliva and health status; 2 of 5 healthy kittens had high concentration of salivary virus, compared with 5 of 5 unhealthy cats (one was a kitten).

Experimental bite study—Seroepidemiologic evidence suggested that biting may be an important mode of FIV transmission. Two of the kittens (No. 3326 and 3327) that were subjected to the bites of cats CJ1 and CJ2 seroconverted to FIV within 37 days, and the remaining kittens (No. 3334 and 3340) seroconverted after 60 days. Virus was successfully reisolated from the blood of all 4 kittens at the time of seroconversion.

Cat-to-human transmission—Human beings (n = 18) that were exposed to FIV, in some instances by relatively high risk occurrences, were uniformly seronegative for FIV when tested by immunoblot analysis (Table 7).

Discussion

The rate of FIV infection among high-risk cats was 14% in the United States and 19% in Canada. These figures are less than half the infection rate for cats in Japan.[12] Possible reasons for these differences have been discussed.[12] The incidence of FIV infection among clinically ill high-risk cats was significantly higher than the incidence of the infection in healthy low-risk cats. The same relationship between FIV infection and illness also has been observed in other

studies[12] and suggests that FIV is pathogenic to cats. Seroepidemiologic observations are important, because the immunodeficiency phase of natural FIV infection has not been experimentally re-created; experimentally infected SPF cats kept in pathogen-free environments developed only a transient primary illness and a sustained carrier state (without clinical signs of disease).[6] The FIV infection rate among clinically ill cats also was somewhat lower than the 30% FeLV infection rate reported in similar groups of chronically ill cats more than a decade ago.[13-15]

The clinical phase of FIV infection was seen most often in cats between 6 and 15 years old, although cats in the age range between <1 year and >15 years old sometimes were also affected. The greater age of FIV-infected cats was in contrast to FeLV-infected cats, which tend to become infected at a rapid rate from kittenhood onward.[16] The older age of the cats at the time of disease development suggests either than infection with FIV develops at a much slower rate than for FeLV or that there is a longer period between initial infection and disease manifestation. The latter scenario is supported by results of experimental inoculation studies.[6] Male cats were apt to be infected with FIV at nearly 3 times the expected rate. Cats that were kept strictly indoors were at the lowest risk, and cats that were kept only outdoors were at the highest risk. The typical cat with FIV-related disease, therefore, was most likely to be >6 years old, male, and free-roaming.

The FIV-infected cats usually were domestic type. Infection of purebred cats was uncommon, and probably was attributable to environmental factors and not to genetic predisposition. Purebred cats were more likely to be kept strictly indoors, which would remove one major risk factor for infection.

The major clinical features of the immunodeficiency-like phase of naturally acquired FIV infection were rather stereotyped and essentially the same as those described for FIV-infected cats in Japan.[2,12] Oral cavity disease, including chronic gingivitis, periodontitis, stomatitis, tonsillitis, and glossitis were seen in 56% of the FIV-infected cats. Gastrointestinal tract disease, usually chronic enteritis or enterocolitis, was seen in 20% of the affected cats. Infections of the upper respiratory tract were seen in more than a third of the cats; however, abnormal CNS signs were seen in only 1%. This latter figure may be low because of the way practitioners selected cats for testing. Central nervous system signs of disease usually were of cortical origin; vague behavioral changes, dementia, changes in excretion/elimination habits, rage, and abnormal movements of the facial musculature have been the most common clinical manifestations in cats of this and other studies.[1,2] Neoplasia, usually lymphosarcoma or myeloproliferative disorders, were seen in 3% of the cats. Lymphosarcoma associated with FIV infection also has been reported in a Japanese study,[12] and 1 kitten that was experimentally infected with FIV developed a myeloproliferative disorder.[6] The number of cats with upper respiratory tract, oral cavity, and gastrointestinal tract diseases reflected the predominance of these disorders in this study population, but the actual proportion of cats with these disorders may be skewed. Original descriptions of cats with naturally acquired FIV infection emphasized such signs, and practitioners obviously were concentrating on cats with such signs of disease for testing. The breadth of diseases associated with FIV infection remains to be elucidated.

Several types of hematologic disorders were seen in FIV-infected cats. Forty-two percent of affected cats were anemic to some degree, and 30% were leukopenic. The leukopenia was associated with neutropenia (22%) and/or lymphopenia (53%). The pathogenesis of the anemia and leukopenia has not been determined.

To gain information about possible modes of FIV infection, 2 other retrovirus infections of cats, FeSFV and FeLV, also were evaluated in the same population. Feline leukemia virus infection is spread by intimate contact between persistently viremic and susceptible cats.[15] The virus is shed in urine, saliva, and feces and is transmitted by ingestion and biting. Outside the cat's body, FeLV is quickly inactivated, so prolonged and intimate contact between infected and noninfected cats is required for efficient transmission. Thus, FeLV infection can become rampant in enclosed multiple cat households and catteries. These types of environments provide the intimate contact that is essential for transmission, and virtually all susceptible cats will become FeLV-infected within 1 to 18 months.[17,18] Given the essential differences in FeLV and FIV transmission, it was not surprising that the 2 infections appeared to be transmitted independently of each other.

In contrast to FeLV and FIV infections, FeSFV and FIV infections often developed together. This finding again was not surprising; FeSFV and FIV infections share many common features. Both viruses are inefficiently transmitted by contact alone,[6,8] and FeSFV, like FIV, can be efficiently spread by bites.[8] As seen in the SPF kittens of this study, a single artificially administered bite can transmit FIV in most instances. The same experiment has been done for FeSFV.[6] The single difference in the apparent modes of transmission for these 2 viruses is that FeSFV infection is commonly transmitted in utero, but FIV is not.[6,8]

Almost all FIV-seropositive cats have isolatable virus in their blood, and most of these shed appreciable amounts of virus in their saliva. Therefore, all FIV-seropositive cats should be considered infectious. On the basis of the 10 cats reported herein, it appears that clinically ill cats shed more virus in their saliva than do healthy ones. It follows that ill FIV-infected cats would be more infectious than would their healthy counterparts.

Epidemiologic evidence further supported the role of cat bites in FIV transmission. Males are almost 3 times more likely to be infected than are females. Male cats are more territorially aggressive than are females and have a disproportionate incidence of cat bite wounds and abscesses. Further, cats that are kept strictly indoors, even in groups, do not usually resort to physical aggression. Given the steady transmission of FIV in nature, distinct epidemiologic risk factors (males, free-roaming), efficiency of experimental bite transmission, and the inefficiency of nontraumatic contact, lactogenic, and in utero transmission,[6] it is reasonable to assume that bites may be the major mode of transmission in nature.

Cats infected with FIV live in close contact with human beings. There is some concern, therefore, about the public health importance of this virus. The fact that FIV is found throughout the world suggests that, in cats, the virus greatly predates the modern human epidemic of acquired immune deficiency syndrome. The cat virus, although morphologically and biochemically similar to HIV, is antigenically distinct.[1,6] Until the virus is genetically sequenced, however, its position in the evolutionary tree of lentiviruses is unknown. A small number of human beings that have had close contact with infected cats have been tested for antibodies to the virus; in no instance were any such antibodies found. Lentiviruses are found in many species of animals, and there appears to be a great deal of species specificity. The human virus, HIV, appears most closely related to SIV.

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Feline immunodeficiency virus infection in cats of Japan

Takuo Ishida, DVM, PhD; Tsukimi Washizu, MS, DVM; Kazushige Toriyabe, MS, DVM; Shigekatsu Motoyoshi, DVM, PhD; Isamu Tomoda, DVM; Niels C. Pedersen, DVM, PhD

Summary: *A seroepidemiologic survey for feline immunodeficiency virus (FIV) infection was conducted in Japan. Between June and December 1987, individual sera (n = 3,323) were submitted by veterinary practitioners from many parts of the country. Specimens were from 1,739 cats with clinical signs suggestive of FIV infection and from 1,584 healthy-appearing cats seen by the same practitioners. The overall FIV infection rate among cats in Japan was 960/3,323 cats (28.9%). The infection rate was more than 3 times higher in the clinically ill cats, compared with that in the healthy cats of the same cohort (43.9 vs 12.4%). Male cats were 1.5 times as likely to be infected as were females. Almost all FIV-infected cats were domestic cats (as opposed to purebred cats).*

Complete clinical history was available for 700 of 960 FIV-infected cats. Of these 700 FIV-infected cats, 626 (89.4%) were clinically ill, and the remainder did not have clinical signs of disease. The mean age at the time of FIV diagnosis for the 700 cats was 5.2 years, with younger mean age for males (4.9 years) than for females (5.8 years). Most of the infected cats (94.7%) were either allowed to run outdoors or had lived outdoors before being brought into homes. The mortality for FIV-infected cats during the 6 months after diagnosis was 14.7%, and the mean age at the time of death was 5.7 years.

Concurrent FeLV infection was seen in 12.4% of the FIV-infected cats, but this was not much different from the historical incidence of FeLV infection in similar groups of cats not infected with FIV. Dually infected cats were of a younger mean age than were cats infected only with FIV (4.3 vs 5.2 years). Cats coinfected with FIV and FeLV tended to die at a younger age than did cats infected only with FIV (4.6 vs 5.7 years). There was no obvious relationship between FIV infection rate and antibody titer interpreted as positive for infection with feline infectious peritonitis virus, and feline infectious peritonitis among FIV-infected cats was less than historically expected for the population as a whole.

The clinical signs seen most frequently in FIV-infected cats were stomatitis/gingivitis (52.4%), chronic upper respiratory tract disease (28.9%), anemia (17.9%), emaciation (14.6%), bacterial infections (14.4%), chronic skin disease (13.4%), and chronic enteritis (11.9%). Less frequently observed clinical findings were renal disease, fever of unknown origin, lymphadenopathy, and leukopenia. A small number of FIV-positive cats had infections caused by opportunistic organisms. These diseases included haemobartonellosis, notoedric mange, toxoplasmosis, cryptococcosis, Pseudomonas infection, candidiasis, and atypical mycobacterial infections. Three young FIV-infected cats had FeLV-negative lymphosarcoma or fibrosarcoma.

Feline immunodeficiency virus (FIV) was first described in 1987.[1] The virus originally was called feline T-lymphotropic lentivirus, but the name has been changed to comply with the international nomenclature for lentiviruses that cause acquired immunodeficiency syndrome in man and animals.[2] The feline virus is morphologically and biochemically similar to the human and simian immunodeficiency viruses, but is antigenically distinct.[1] Shortly after the discovery of FIV in the United States, an identical virus was isolated from cats in Japan[3] and the United Kingdom.[4] These latter studies reconfirmed the close relationship between FIV infection and a naturally acquired immunodeficiency-like syndrome in FeLV-negative cats.

The study reported here is an extension of earlier work on FIV infection in Japan,[3] and involved 3,323 cats that were tested during a 6-month period between June and December of 1987. Information gained from this extended survey revealed the magnitude and clinical expression of FIV infection in cats of Japan. It also supported previous studies that link FIV infection with a number of chronic infectious disease syndromes of cats.

Materials and methods

Source of sera—Japanese veterinarians were encouraged to submit sera from cats with signs of chronic infections and sera from healthy-appearing

From the Departments of Clinical Pathology (Ishida, Washizu, Toriyabe) and Medicine (Motoyoshi, Tomoda), School of Veterinary Medicine, Nippon Veterinary and Zootechnical College, 1–7–1 Kyonan-cho, Musashino-shi, Tokyo 180, Japan, and the Department of Medicine, School of Veterinary Medicine, University of California, Davis, CA. 95616 (Pedersen).

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cats living in the same households or treated by the same practitioners. Sera (n = 3,323) were submitted for testing during the period between June and December of 1987. Of 960 FIV-positive cats, 700 with reasonably complete clinical history were selected for further review.

Antibody assays—Antibody assays for FIV were conducted by the Department of Clinical Pathology, Nippon Veterinary Zootechnical College. An indirect fluorescent antibody (IFA) assay was used as a screening test for FIV antibodies. The substrate for the IFA assay was FIV-infected feline T lymphocytes.[1] Blood mononuclear cells were purified from the blood of specific pathogen-free cats by use of sodium diatrizoate[a] density-gradient centrifugation. The cells were cultured in RPMI-1640 medium containing 10% fetal bovine serum and were stimulated with 5µg of concanavalin A[a]/ml. Three days later, the cultures were treated with 100 U of human recombinant interleukin-2[b] (rIL-2)/ml. The resultant T-lymphocyte culture was infected with the Petaluma strain of FIV.[1] Serum samples were tested at a dilution of 1:10, and those that were positive by results of IFA assay were confirmed to be positive by immunoblot analysis, using electrophoresed FIV proteins.[3]

FeLV and feline infectious peritonitis virus (FIPV) assays—The FeLV-p27 antigen in sera was detected by an ELISA that used monoclonal antibodies to this protein.[5,6] Serum antibodies to FIPV also were determined by ELISA.[7]

Virus isolation—Blood lymphocytes from healthy and clinically ill FIV antibody-positive cats were isolated and established as replicating cell lines as previously described.[1] The cultures were maintained by adding fresh cells, rIL-2, and medium at 3- to 10-day intervals. Cultured cells were examined periodically for a characteristic cytopathic effect (CPE).[1] When CPE was evident, the culture fluid was examined for Mg^{++}-dependent reverse transcriptase activity, and pelleted material was tested by immunoblot analysis against FIV-positive human sera.[1]

Statistical methods—To examine the relationship between FIV serostatus and the variables of age, breed, sex, and health and environment statuses, multiway and 2 × 2 tables were generated by use of a computerized statistical program.[8] The relationship of each variable was evaluated, using the Pearson χ^2 test.

Results

Among the 3,323 sera that were tested, 906 (28.9%) were positive for FIV antibody (Table 1). The FIV infection rate was significantly ($\chi^2 = 401.9$; $df = 1$; $P << 0.005$) higher in cats that were clinically ill (764/1,739; 43.9%) than in the healthy cat population (196/1,584; 12.4%). The male to female ratio of 2.3:1 in FIV-infected cats was significantly ($\chi^2 = 56.5$; $df = 1$; $P < 0.005$) greater than the 1.5:1 ratio seen in the group as a whole. Domestic cats were more likely to be infected than were purebred cats (898/2,627 vs 62/696). The predisposition of domestic cats to FIV infection was statistically significant ($\chi^2 = 169.1$; $df = 5$; $P << 0.005$). Cats kept strictly indoors (16/236; 7%) were significantly ($\chi^2 = 79.5$; $df = 3$; $P < 0.005$) less likely to become infected than were cats that were allowed to roam outside (246/657; 37%). There was a significant ($\chi^2 = 394.2$; $df = 4$; $P << 0.005$) increase in the FIV infection rate with age (Table 1). This was most pronounced for the first 10 years of life.

Table 1—Vital statistics of 3,323 cats in Japan that were surveyed for feline immunodeficiency virus (FIV) infection

Vital statistic	Total surveyed	No. seropositive for FIV (%)
Health status		
Healthy	1,584	196 (12)
Clinically ill	1,739	764 (44)
Sex distribution		
F	1,330	288 (22)
M	1,993	672 (34)
Age distribution (years)		
≤1	690	28 (4)
1 to 5	1,871	545 (29)
6 to 10	612	328 (54)
11 to 15	135	45 (33)
≥15	15	7 (47)
Breed distribution		
Domestic	2,627	898 (34)
Siamese	171	30 (18)
Persian	193	16 (8)
Himalayan	260	12 (5)
Abyssinian	36	4 (11)
Others	36	0 (0)
Environmental status		
Indoor (owned)	236	16 (7)
Outdoor (owned)	165	65 (39)
Indoor/outdoor (owned)	437	159 (36)
Outdoor (unowned)	55	22 (40)

Seven hundred FIV-infected cats with reasonably complete clinical history were selected for further study. The vital statistics for this group of 700 cats are given in Table 2. Among 488 FIV-infected male cats, 415 (85%) were sexually intact; 172 of 212 (81.1%) FIV-infected female cats also were unneutered. The incidence of FIV infection in sexually intact vs neutered cats was not significantly different, however, from the proportion of neutered vs unneutered cats in a comparable group of FIV-negative cats; 379 of 450 (84.2%) FIV-negative males and 236 of 300 (78.7%) FIV-negative females were sexually intact. Greater than 95% of the FIV-positive cats were domestic shorthairs. The age of FIV-infected cats at the time of diagnosis ranged from 2 months to 18 years (Fig 1). The mean (± SD) age was 4.9 ± 2.8 years for males and 5.8 ± 3.8 years for females. The overall mean age was 5.2 ± 3.2 years. Almost all (94.7%) FIV-infected cats were

[a]Hypaque 1077, Sigma Chemical Co, St Louis, Mo.
[b]rIL-2, Cetus Corp, Emeryville, Calif.

*Table 2—Vital statistic ratios for 700 cats that were naturally infected with feline immunodeficiency virus (FIV)**

Vital statistic	No. of cats (%)	Ratio
Healthy status		
Clinically ill	626 (89)	8:1
Healthy	74 (11)	
Sex distribution		
M	488 (70)	2:1
F	212 (30)	
Neuter status		
Sexually intact	587 (84)	5:1
Neutered	113 (16)	
Breed distribution		
Domestic	666 (95)	19:1
Purebred	34 (5)	
Environmental status		
Outdoor/indoor	663 (95)	19:1
Strictly indoor†	37 (5)	
FeLV serostatus		
Negative	613 (88)	7:1
Positive	87 (12)	

*Over a 6-month period, mortality in this group of FIV-infected cats was 14.7%. Age of cats ranged from 2 months to 18 years, with mean value of 5.2 years; mean age of males and females was 4.9 and 5.8 years, respectively. † Of these 37 cats, 25 lived in multiple-cat households in which indirect contact with other outdoor/indoor cats was possible.

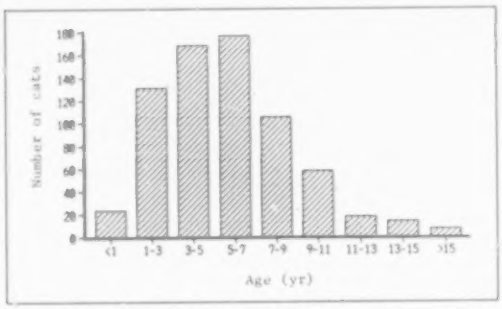

Figure 1—Distribution by age of a select group of FIV-infected cats (n = 700).

either outdoor or indoor/outdoor-roaming at the time of diagnosis, or had been moved strictly indoors from such environments earlier in life. Of the 37 cats that had been kept strictly indoors, 25 lived in multiple-cat households where contact with other outdoor/indoor cats was possible.

Most (626/700) of the FIV-infected cats were clinically ill at the time of diagnosis. The mean age of FIV-infected clinically ill cats (5.3 ± 3.1 years) was somewhat greater than that of healthy FIV-infected cats (4.2 ± 3.2 years). The diseases in the FIV-infected cats were variable in severity and nature. More than half (54.7%) of the 700 FIV-infected cats had multiple clinical signs of disease; the mean number of signs per cat was 1.9 (Fig 2). Table 3 gives the most frequent clinical manifestations of FIV-infected cats. Mild disorders included chronic conjunctivitis, rhinitis, and low-grade fever of unknown origin. Severely ill cats usually had multiple problems such as stomatitis, chronic upper respiratory tract disease, anemia, enteritis, and emaciation. Chronic stomatitis/gingivitis was the most frequent (52.4%) complaint in the 700 cats. This was followed in frequency by chronic upper respiratory tract disease (28.9%), anemia (17.9%), emaciation (14.6%), unspecified bacterial infections (14.1%), chronic skin infections (13.4%), and enteritis (11.9%). Additional but less frequently observed signs included renal disease, fever of unknown origin, lymphadenopathy, and leukopenia.

A number of the 700 FIV-infected cats with clinical history of good health suffered from infections that could be considered opportunistic in nature. These infections included haemobartonellosis (n = 10), chronic bacterial pneumonia (n = 7), generalized notoedric mange (n = 6), feline infectious peritonitis (n = 5), pyothorax (n = 5), toxoplasmosis (n = 3), severe intestinal coccidiosis (n = 3), systemic candidiasis (n = 2), and atypical mycobacteriosis (n = 1). Other FIV-infected cats had less specific clinical diagnoses such as hepatitis (n = 10), ascites (n = 8), or abortion (n = 4). Three FIV-infected cats had lymphosarcoma, and 1 had periosteal fibrosarcoma. Of these latter cats, 3 were between 4 and 5 years old and were sero-negative for FeLV antigen and antibody.

Mortality among FIV-infected cats during the 6-month study was 103 of 700 (14.7%). The mean age at death was 5.7 ± 2.9 and 5.9 ± 4 years in males and females, respectively. Clinical signs of disease frequently associated with fatality were stomatitis (49.5%), anemia (45.6%), emaciation (32%), renal disease (32%), and chronic upper respiratory tract disease (30%).

Concurrent FeLV infection was seen in 87 of 700 (12.4%) FIV-infected cats; most (83.9%) of these

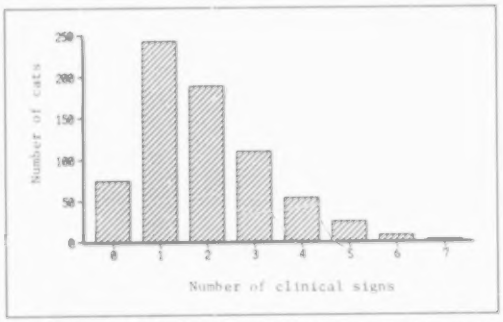

Figure 2—Distribution of FIV-infected cats according to the number of clinical signs at the time of diagnosis (n = 700).

Table 3—Common clinical abnormalities observed in 700 cats infected with feline immunodeficiency virus in Japan

Clinical signs	No. of cats (%)
Stomatitis/gingivitis	367 (52.4)
Chronic upper respiratory tract disease	202 (28.9)
Anemia	125 (17.9)
Emaciation	102 (14.6)
Bacterial infections (undefined)	99 (14.1)
Chronic skin disease (undefined)	94 (13.4)
Chronic diarrhea	83 (11.9)
Renal failure	65 (9.3)
Fever of unknown origin	60 (8.6)
Lymphadenopathy	56 (8.0)
Leukopenia	35 (5.0)

cats were male. The mean age of FeLV-positive, FIV-infected cats was 4.3 ± 2.2 years, compared with 5.3 ± 3.2 years for their FeLV-negative counterparts. Feline leukemia virus infection was seen in 20 of 103 (19.4%) FIV-infected cats that died; dually-infected cats tended to die at a younger mean age than did FeLV-negative FIV-infected cats (4.6 ± 2.4 vs 5.9 ± 3.4 years).

Feline infectious peritonitis titer was determined for 415 of the 700 FIV-infected cats (Fig 3). The mean FIPV antibody titer in these 415 cats was 1:300. The mean antibody titer and titer distribution in the 415 FIV-infected cats was the same as those values in a comparable number of FIV-negative cats that also were tested for FIPV (data not shown). There were 5 cats with confirmed FIP among the 700 FIV-infected cats.

Virus isolation was attempted in 29 clinically ill and 3 healthy FIV antibody-positive cats. All but one lymphocyte culture had CPE beginning 1 to 3 months after culture initiation. The 31 cell cultures that developed CPE were confirmed positive for FIV by results of IFA assay, reverse transcriptase expression, and FIV-p28 antigen detection by immunoblot analysis.

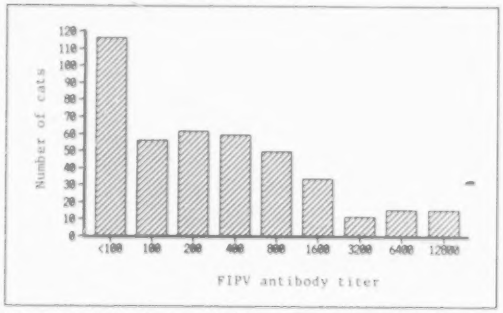

Figure 3—Distribution of feline infectious peritonitis virus (FIPV) antibodies by titer in cats that were naturally infected with FIV (n = 415).

Discussion

The overall FIV infection rate among 3,323 cats that were tested was nearly 30%, which was significantly higher ($P<0.005$) than the FIV infection rate in a similar study group from the continental United States and Canada.[9] Because the study did not involve cats selected at random, it was not possible to determine the infection rate among cats in Japan at large. The overall infection rate in the general population probably would be lower than the 12.4% incidence that was observed in the healthy cats in the test population of this study. It is unlikely to be as low, however, as the 1.5% infection rate in a group of healthy cats from the United States.[9]

The significantly higher FIV infection rate in cats of Japan, compared with those of the United States and Canada, deserves mention. The cat is a more popular pet in Japan than in North America and the human population density in Japan is far greater. There are more cats per given area in Japan, therefore, than in the United States and Canada. Most pet cats in Japan are kept by people that live in housing with yards; about 95% of pet cats in Japan are allowed to roam free. The number of cats kept by occupants of high-rise tenements is relatively low, compared with that in the United States. Free-roaming has been found to be a high-risk factor for FIV infection in cats of this and other studies.[9] High cat density would further increase the spread of infection, because it would put susceptible and infected cats in greater contact with each other. It also may increase the level of territorial and sexual aggression (biting, scratching) because many more cats would share the same terrain.

Bites have been implicated as a major mode of FIV transmission.[9,10] Biting among cats is usually associated with territorial aggression among freely roaming populations. Biting is a behavior that is observed infrequently among cats that are kept strictly indoors. Biting also has been associated with sexual activity among outdoor cats. If biting and sexual aggression were strongly linked, and if biting were a major mode for FIV transmission, sexually intact cats presumably would have higher incidence of FIV infection than would neutered cats. Sexual intactness did not appear to be a risk factor for cats in Japan, however. The rate of FIV infection was the same in intact and neutered cats. The fact that neutering did not influence the FIV infection rate among cats in Japan also suggested that FIV is not transmitted venereally to any extent.

The mean age of FIV-infected male cats at the time of diagnosis was 4.9 years, which was 1 year younger than the age for females. This suggested that male cats were infected more rapidly than were females. Male cats in Japan were also 1.5 times more likely to be infected than were females. It appears, therefore, that male cats were more likely to engage in activities that put them at increased risk. Male cats are more aggressive than female cats and are more likely to be treated for bite-related disorders (wounds, cellulitis, abscesses) than are females, again suggesting that biting is a major mode of FIV transmission. If FIV were transmitted only by normal nontraumatic contact or by blood-sucking vectors such as fleas, both genders should be at similar risk, and indoor cats also would have a higher infection rate.

The period between infection and the development of the acquired immunodeficiency-like phase of infection was not determined, but it appeared to be long. Clinically ill FIV-infected cats had a mean age that was 1 year greater than that of FIV-infected cats that were healthy, indicating that the period between infection and disease was >1 year. Socializing among cats begins at ages as early as 4 months, and exposure would be expected within the first

few years of life. The fact that many clinically ill FIV-infected cats were aged at the time of illness detection suggested that years can pass between the time of infection and illness. This assumption is supported by other studies of naturally and experimentally induced FIV infection in relatively closed populations of cats.[1,9] The mortality among clinically ill FIV-infected cats appeared to be slow in developing. Only 14.7% of the clinically ill cats died within 6 months of diagnosis. This observation also was in agreement with other studies of the naturally acquired disease.[1]

The diseases in clinically ill FIV-infected cats in Japan were virtually identical to those observed in similar groups of cats in the United States.[1,9] Stomatitis, peridontitis, and gingivitis attributable to chronic oral infections were seen in >50% of the cats. In our experience, in Japan, approximately 50% of cats evaluated for chronic oral cavity infections turned out to be FIV infected. Oral infections apparently are sensitive indicators of immunosuppression in cats. Interestingly, oral cavity disease may have an important role in the transmission of FIV. Saliva might contain more virus if the oral lesions are exudative and bleeding, and this might greatly enhance the efficacy of bite-related transmission. This is supported by results of studies of experimentally induced FIV infection.[9,10]

Opportunistic infections were recognized in a few FIV-infected cats. They included haemobatonellosis, generalized notoedric mange, cryptococcosis, candidiasis, pyothorax, toxoplasmosis, severe intestinal coccidiosis, bacterial pneumonia, *Pseudomonas* infections, and atypical mycobacteriosis. Such infections were considered highly indicative of underlying immunodeficiency, and many of them are included in the diagnosis of acquired immunodeficiency syndrome in human beings.[11,12] In this group of cats, the low incidence of neoplasms that normally are associated with FeLV also was interesting. Two cats with lymphosarcoma and one with fibrosarcoma were relatively young FIV-infected cats with no evidence of either ongoing or previous FeLV infection.

Concurrent FeLV infection was seen in about 12% of FIV-infected cats. This may not be a significant relationship, however, because the overall FeLV infection rate in similar groups of cats in Japan is around 10%.[13] A lack of relationship between FeLV and FIV infections also was seen in a group of cats from the United States and Canada.[9]

The incidence of FIPV antibodies was the same in FIV-infected and FIV-noninfected cats, indicating that coronavirus and FIV infections were not linked. The incidence and titer of FIV antibodies in the group of cats reported herein corresponded with those previously described for cats in Japan.[14] The frequency of clinical feline infectious peritonitis in the cats of this study (5/700 or 0.7%) was smaller than the expected frequency (4.9%) of the disease previously reported in the cat population of Japan.[14]

Mortality in FIV-infected cats during the 6-month study was 14.7%, which was much less than the 54% mortality observed for FeLV-infected cats in Japan during a 14-month period.[13]

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Economic Note

Trend in real starting salaries of US veterinary medical college graduates, 1980-1988

Average starting salaries of US veterinary medical graduates have been reported annually since 1980.[1-4] The average starting salary increased 29.1%, from $17,175 in 1980 to $22,181 in 1988 (Table 1). This represents a compounded annual growth rate of 3.2%.

Analysis by type of employer showed that the average starting salary in private practice increased 24.4%, from $18,552 in 1980 to $23,079 in 1988 (Fig 1). In nonprivate practice, the average starting salary increased 43.8%, from $17,967 in 1980 to $25,832 in 1988. The average starting salary for graduates pursuing advanced study increased 31%, from $12,033 to $15,766.

These data show that graduates' starting salaries have risen in money terms. In real terms, ie, correcting for the increased cost of living, graduates' average starting salary declined. The most widely used measure of the change in the cost of living at the retail level is the Consumer Price Index for all urban consumers. The Consumer Price Index increased 43.4% from 1980 to 1988.

Analysis of graduates' starting salaries in real terms was accomplished by expressing the average starting salaries for each type of employer for each year, 1980 through 1988, in 1983 dollars. Table 1 shows average starting salaries in money terms and in real terms (1983 dollars).

Results show that, in real terms, graduates' average starting salary declined from $20,768 in 1980 to $18,699 in 1988, a decrease in purchasing power of 10% over the 8-year period. In private practice, the average real starting salary declined 13.3%, whereas in nonprivate practice, the average real starting salary increased 0.2%, or virtually remained the same. Starting incomes for graduates enrolled in

Prepared by J. Karl Wise, PhD, Wisemark Associates, PO Box 681189, Schaumburg, IL 60168-1189.

Table 1—Real starting salaries of US veterinary medical graduates, 1980–1988

Year	Money income ($)				Real income (1983 dollars)			
	Private practice	Nonprivate practice	Advanced study	All graduates	Private practice	Nonprivate practice	Advanced study	All graduates
1980	18,552	17,907	12,033	17,175	22,433	21,725	14,550	20,768
1981	18,832	18,059	12,937	18,314	20,626	19,780	14,170	20,059
1982	19,351	20,864	13,781	18,700	19,970	21,531	14,222	19,298
1983	19,872	22,865	13,541	19,194	19,872	22,865	13,541	19,194
1984	20,237	21,493	13,797	19,527	19,403	20,607	13,228	18,722
1985	20,956	21,814	14,199	20,164	19,405	20,199	13,148	18,672
1986	21,205	22,027	15,186	20,478	19,275	20,022	13,804	18,615
1987	22,152	25,697	15,272	21,693	19,427	22,536	13,394	19,025
1988	23,079	25,832	15,766	22,181	19,455	21,776	13,291	18,699*

*Based on projected 4.02% increase in cost of living for 1988.

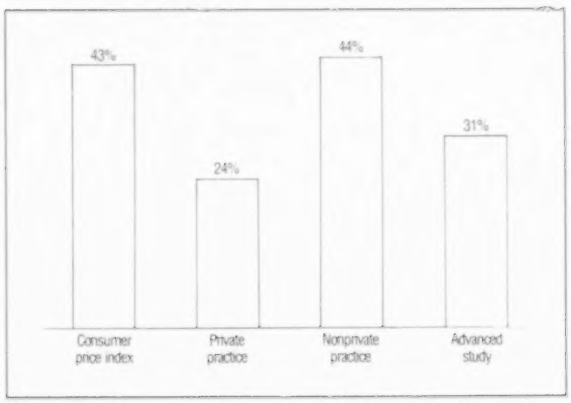

Figure 1—Percentage increase in starting salaries of US veterinary medical college graduates, 1980-88.

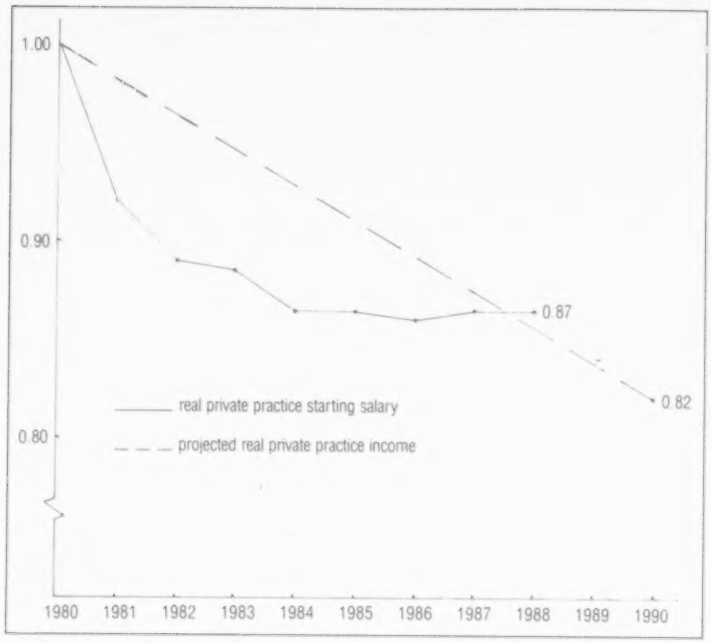

Figure 2—Standardized trend in real starting salaries (1980 = 1.0) in private veterinary practices.

advanced study declined 8.6% in real terms.

On the basis of the 1985 AVMA manpower study, average real private practice net individual income was projected to decline by the year 2000 to 75% of its value in the year 2000.[5-6] Most of the decline was projected to occur by 1990, as it was thought average real private practice income would fall to 82% of its 1980 level by the year 1990. From 1980 to 1990, this represents an 18% decline in projected real income. Intuitively, the impact of the projected decline would be reflected in depressed entry-level salaries, followed by lower overall average real incomes in subsequent years.

Data presented here suggest that, to date, at least for entry-level positions in private practice, the decline in real private practice income has not been as severe as earlier projected. Expressed in standardized form (1980 = 1.0), Figure 2 shows the decline in average real private practice income projected in the 1985 AVMA manpower study. In 1990, it was projected that average real private practice income would be 82% of its 1980 level. For comparison, Figure 1 also shows the decline in average real private practice starting salary. In 1988, the average real starting salary in private practice was 87% of the 1980 average real starting salary. Interestingly, real private practice starting salaries have been flat since 1984, revealing that 1988 graduates have neither gained nor lost purchasing power relative to that for 1984 graduates.

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